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AUTHORS ALONE ARE RESPONSIBLE FOR OPINIONS EXPRESSED IN THEIR CONTRIBUTIONS

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BULLETIN OF
THE NEW YORK ACADEMY
OF MEDICINE

AUGUST 1944

JAUNDICE FOLLOWING ADMINISTRATION OF HUMAN SERUM

Harvey Lecture, March 16, 1944

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JAUNDICE following administration of materials containing serum of the homologous species has been observed repeatedly both in man and in lower animals. It is still unknown whether this type of jaundice is identical with naturally occurring socalled infectious hepatitis.

One of the earliest reports of this condition is that of Hirsch[1] who records an outbreak in 1883-4 of jaundice among individuals vaccinated with "humanized lymph in glycerine." Among 1,289 persons vaccinated, 191 or 14.8 per cent developed jaundice after incubation periods "extended to several weeks and even to a couple of months." No cases developed among 500 person vaccinated with a different lymph.

In 1918 Theiler[2] reported a condition known as "staggers" in horses, which followed administration of homologous serum. Jaundice was a marked sign. The incubation period varied from 27 to 165 days and the mortality varied from 4 to 18 per cent among large groups of immunized animals. Slagsvold[3] found that 101, or 4.2 per cent of 2,400 horses treated with anti-anthrax serum developed liver damage from 8 to 95

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days following injection of the material. In most cases the elapsed period was 50 to 60 days. A similar condition in horses following the administration of equine encephalomyelitis vaccine containing homologous serum has been observed in this country.[4, 5]

MacNalty[6] drew attention to the occurrence of jaundice among 37 of 82 to 109 persons who had been given convalescent measles serum from the same pool of material. Seven deaths were recorded. Convalescent measles serum in doses of 4.5 cc. was given to seven children and in 75 to 83 days they all developed severe jaundice. Three cases terminated fatally. Two months later two children who had had contact with these patients developed mild infective hepatitis.[7]

Findlay and MacCallum[8] found that hepatitis was a complicating factor in the use of yellow fever vaccines. Different types of vaccines used produced jaundice; the only common components in the products were human serum and the attenuated virus of yellow fever. During a five-year period 3,100 persons were immunized against yellow fever and 89, or 2.87 per cent, of these developed jaundice. The average incubation period was 2 to 3 months with a range of 36 days to 7 months. Hepatitis has been associated with the use of yellow fever vaccine by other workers. Soper and Smith[9] described the first series of cases occurring in South America. A vaccine containing immune monkey serum and tissue culture virus was used. Among 244 persons immunized, 66, or 27 per cent, developed hepatitis. The incubation period was prolonged.

Fox and his coworkers[10] studied two outbreaks of hepatitis following the use of yellow fever vaccine in Brazil. In 1939, 304 persons were immunized and 27 per cent suffered from hepatitis during the fourth and fifth months following immunization. In 1940, 35 lots of vaccine were used to vaccinate 107,169 persons. There were only 93 cases, or 0.1 per cent, of jaundice among 87,878 persons given material from 33 lots of vaccine. Two other lots produced a greater amount of jaundice among the recipients. The attack rate with one lot used among 9,604 individuals was 7.68 per cent and for another lot given to 9,587 persons the rate was 1.56 per cent. The average incubation period was 24.8 weeks for the 33 lots of vaccine, 17.8 weeks for the lot producing the highest attack rate of icterus, and 20.4 weeks for the other lot. A total of 25 deaths was recorded.

Recent U. S. Army experience with yellow fever vaccine resulted

in 28,585 cases of hepatitis with 62 deaths as of July 24, 1942.[11]

Immunization against pappataci fever has also resulted in the appearance of jaundice after a prolonged period. The method used for immunization consisted of separate inoculations of virus and antiserum. The source of virus was human blood obtained during the acute stage of the disease. Sergiev *et al*,[12] studied 100 cases of jaundice resulting from the use of such materials. The incubation period varied from 63 to 146 days; about 50 per cent of the cases occurred between 85 and 95 days after inoculation. About 30 per cent of those vaccinated subsequently developed hepatitis.

A report of the British Ministry of Health[13] attributes 48 cases of jaundice with 8 deaths to the use of pooled convalescent and adult human serum for measles. Incubation periods ranged from 16 to 161 days. Twelve cases of jaundice due to transfusion were also recorded. A number of other cases of hepatitis developed following the use of mumps convalescent serum. The writers conclude that, "any doubt as to the reality of the association is removed by the frequency with which hepatitis has followed the injection of human blood products."

In addition to the reports of jaundice secondary to immune sera and supposedly normal serum, there were in 1943 two reports of jaundice following the administration of pooled human plasma, reconstituted dried human serum and blood transfusion. Beeson[14] in this country reported seven cases. Of these, four had received one or more transfusions of citrated blood only. The other three persons had received both pooled plasma and citrated blood at different times. In five of the cases the minimum possible incubation period varied from 69 to 111 days. In England, Morgan and Williamson[15] saw nine cases which developed following transfusion with serum, plasma or both. The incubation periods varied from 7 to 16 weeks.

Findlay and Martin[16] present evidence that an "infective icterogenic agent" is present in the nasopharynx of individuals developing hepatitis following administration of yellow fever vaccine. While some doubt might be maintained concerning two of the cases produced by this experimental approach, the evidence in the third case (Case 2) seems convincing. Voegt[17] attempted to transmit the agent of infective hepatitis from one person to another with results which are suggestive.

Lainer[18] carried out a human inoculation experiment in which he inoculated healthy persons with blood or duodenal juice from patients

three to ten days after the appearance of jaundice. The duodenal juice was introduced into the duodenum by tube and blood was given by transfusion. The study was stated to be entirely negative; duration of observation was not given.

The relation between jaundice following administration of homologous serum and the disease known as infectious hepatitis or epidemic catarrhal jaundice is not known. According to the work of Cullinan,[19] Pickles,[20] and others, infectious hepatitis is spread by droplet infection and has an incubation period varying from 20 to 40 days. Two epidemics, one in Canada, the other in Sweden, reported by Fraser[21] and Hallgren[22] appear to have been due to contamination of drinking water by sewage; transmission by the respiratory tract was not seen. Very few secondary cases have been attributed to association with cases of jaundice following the administration of homologous serum. This disease is further peculiar in that the incubation period is prolonged considerably over that of infectious hepatitis.

Many attempts to isolate the causative agent of infectious hepatitis and serum-induced jaundice have been made, so far without success. It is generally supposed that it is a virus.

Isolation of an etiologic virus was claimed by Siede and Meding[23] who inoculated the chorioallantois of chick embryos with duodenal juice obtained from a jaundiced patient. Proof of isolation consisted only in the ability of their material to kill the chick embryos in 4-5 days. The lethal agent was lost on the fourth transfer.

An opportunity to study an epidemic of jaundice following the use of yellow fever vaccine presented itself when an outbreak occurred in the Virgin Islands in the summer of 1942. Sufficient epidemiological evidence[24] was accumulated to establish the identity of the disease and material was obtained for study. Since August 1942 a human inoculation study has been carried on in an effort to gain further information concerning the nature and etiology of this condition.

Epidemiology

During 1942 a total of 11,358 individuals on the islands of St. Thomas and St. John, Virgin Islands, was inoculated with lot 331 yellow fever vaccine containing pooled human serum. According to reliable data there were 11,265 persons living on the island of St. Thomas and 765 on the island of St. John, but owing to wartime increases in

TABLE I

DATES OF IMMUNIZATION AND NUMBERS OF PERSONS VACCINATED WITH YELLOW FEVER VACCINE IN THE VIRGIN ISLANDS

Date immunized, 1942	*Place*	*Number immunized*	*Date immunized, 1942*	*Place*	*Number immunized*
Mar. 4	St. Thomas	490	Mar. 16	St. Thomas	1,554
Mar. 5	do	403	Mar. 18	do	1,505
Mar. 6	do	624	Mar. 20	do	2,568
Mar. 9	do	1,134	Mar. 23	do	298
Mar. 10	do	392	Mar. 28	do	410
Mar. 11	do	596	Apr. 17	St. John	211
Mar. 12	do	597			
Mar. 13	do	576	Total		11,358

population these figures may be too low. There were 11,147 persons vaccinated with yellow fever vaccine on St. Thomas between March 4 and March 28, 1942, and 211 persons vaccinated on St. John on April 17, 1942. The same lot of vaccine was used throughout the immunization procedure. The dates of vaccination together with the numbers of persons vaccinated are given in Table I.

Jaundice was first noted in May, and by June 2, 1942, about 50 cases had been observed. During the next two weeks it was estimated that between 300 and 500 cases occurred. In order to obtain some exact knowledge concerning the incidence of the disease following the administration of vaccine, a survey was done in the city of Charlotte Amalie, St. Thomas, between July 6 and July 16. A group of 1,198 persons was studied. This sample is roughly 10 per cent of the population involved. The data obtained from this survey are included in Table II. It was established that 14.7 per cent of the vaccinated individuals developed symptoms of hepatitis following vaccination and that the incidence was greatest in the age groups between 20 and 59 years. Among 159 persons who were said not to have been vaccinated, 3 cases occurred. Inasmuch as the vaccination records were not entirely adequate it is somewhat difficult to assess the significance of these cases.

Conditions were not suitable for accurate determination of the incubation period. Among a group of 75 patients from whom reliable and

TABLE II

SAMPLE SURVEY OF CHARLOTTE AMALIE, VIRGIN ISLANDS, SHOWING INCIDENCE OF JAUNDICE FOLLOWING YELLOW FEVER IMMUNIZATION

Age	*Vaccinated population*			*Unvaccinated Population*			*Total*		
	Number Surveyed	*Number of cases of hepatitis*	*Per cent with hepatitis*	*Number Surveyed*	*Number of cases of hepatitis*	*Per cent with hepatitis*	*Number Surveyed*	*Number of cases of hepatitis*	*Per cent with hepatitis*
Under 1	31	0	7.5	17	....	....	297	20	6.7
1—4	127	11		13	....				
5—9	107	9		2	....				
10—14	120	16	11.8	3	....	....	260	29	11.3
15—19	125	13		12	....				
20—24	119	19	18.8	16	....	....	236	40	16.9
25—29	88	20		13	1				
30—34	76	20	21.1	11	....	....	153	28	18.3
35—39	57	8		9	....				
40—44	45	8	22.8	6	....	....	77	16	20.8
45—49	25	8		1	....				
50—54	48	9	21.4	4	....	....	85	15	17.7
55—59	22	6		11	....				
60—64	18	2	12.2	13	1†	....	90	8	8.9
65—69	19	2		10	....				
70+	12	2		18	1				
	1,039	153*	14.7	159	3	1.9	1,198	156	13.0

* Including 12 cases with all signs and symptoms except icterus.
† 1 case with all signs and symptoms except icterus.

observed data could be elicited the average period between immunization and development of hepatitis was 103 days with a range of 75 to 130 days.

The disease was similar to that noted by other observers. Clinically, the disease varied considerably from very mild to extremely severe cases. Onset usually began with headache, pains in the shoulders and back, and frequently with pains in the fingers. A sensation of fullness in the epigastrium was quite characteristic and with this was associated anorexia and nausea. Weakness was a common complaint. Within a day or so the urine was noted to be very dark and within 2 to 3 days icterus of the sclerae appeared. Constipation and clay-colored stools were noted

TABLE III

DATA CONCERNING VIRGIN ISLANDS SERUMS USED IN POOL FOR EXPERIMENTAL GROUPS 2 AND 6

Number	*Date vaccinated, 1942*	*Date jaundiced, 1942*	*Date bled, 1942*	*Results of quantitative van den Bergh test*	*Remarks*
				(mg. per 100 cc.)	
6	Mar. 4	June 22	July 6	0.68	.....
11	Mar. 16	June 28	do	1.07	.....
30	Mar. 4	....	July 8	0.62	.....
40	Mar. 20	....	do	0.56	.....
43	Mar. 6	....	do	1.16	.....
47	Mar. 6	July 7	do	0.84	.....
49	Mar. 4	....	do	0.14	Later developed jaundice.
52	Mar. ?	June 18	do	...	Icteric.
SJA	Apr. 17	July 14	July 17	...	do

during the period of jaundice. Vomiting also occurred and varied considerably in severity. Usually vomiting was limited to one or two episodes but in a few instances was persistent, leading to marked dehydration.

There were cases which presented only dark urine, anorexia, vomiting, headache, and pains with no frank jaundice, while other cases remained jaundiced for at least a month. The average individual was jaundiced for about 6 to 10 days. The degree of illness did not appear to be closely associated with the duration of icterus as many who were jaundiced for a considerable period were ambulatory throughout the illness, while some who were jaundiced only a few days were bedridden during the period.

It seems evident from the standpoint of the previous history of immunization, the prolonged incubation period, and clinical symptoms that the disease under observation was identical with that previously described and designated as homologous serum jaundice.

Under the conditions of the outbreak it was deemed wise to limit our collection of possible infectious material to blood or serum which

TABLE IV

SUMMARY OF GROUPS INOCULATED SHOWING INCIDENCE OF JAUNDICE

Group	*No. in Group*	*Inoculum*	*Cases of jaundice*	
			Number	*Per cent*
1	50	Group 1. Lot 331. Yellow fever vaccine in recommended dose. This vaccine contained pooled human serum and is of the same lot which produced jaundice in the United States Army and in the Virgin Islands.	12	24
2	10	Group 2. Pooled serum collected from nine individuals in the Virgin Islands who had received Lot 331 vaccine. Serum was diluted 1:5. Dose 0.5 ml. S.C.* See Table III.	2	20
3	10	Group 3. Lot 367. Dried yellow fever vaccine containing human serum. Heated in 56° C. water bath for 30 minutes before dilution. Given in recommended dose.	2	20
4	10	Group 4. Pooled weekly specimens of serum from a mild case of jaundice in Group 1. Serum dilution 1 : 3. Dose 0.5 ml. S.C.	0	0
5	10	Group 5. Pooled weekly specimens of serum from a mild case of jaundice in Group 2. Serum dilution 1 : 3. Dose 0.5 ml. S.C.	0	0
6	20	Group 6. Pool of same specimens used in Group 2. Serum dilution 1 : 3. Dose 0.5 ml. S.C. See Table III.	6	30
7	20	Group 7. Pooled weekly serum specimens from a severely jaundiced patient in Group 1. Dilution 1 : 3. Dose 0.5 ml. S.C.	3	15
8	20	Group 8. Pooled weekly serum specimens from a moderately jaundiced patient in Group 1. Dilution 1 : 3. Dose 0.5 ml. S.C.	1	5
9	10	Lot 367. Yellow fever vaccine diluted as recommended and heavily irradiated with ultraviolet light, 1 hour at 2650 A. and 1½ hours at 2537 A.	0	0
10	14	Pooled weekly serum specimens taken before appearance of jaundice from patient in Group 1. Serum dilution 1 : 3. Dose 0.5 ml. S.C.	4	28.7
11	15	Group 11. Single serum specimen from same individual contributing to Group 10. Specimen taken about 2½ months after jaundice had subsided. Serum dilution same as in Group 10. Dilution 1 : 3. Dose 0.5 ml. S.C.	0	0
12	10	L. J. Serum. Had contributed serum to pool used in icterogenic vaccine. Had history of jaundice 1 year previously. Dose 0.5 ml. 1 : 4 S.C.	0	0
13	10	M. B. Serum. Description for Group 12. Serum applies here also. Dose 0.5 ml. 1 : 4 S.C.	0	0

(*Continued on p. 437*)

TABLE IV—(*Continued*)

Group	*No. in Group*	*Inoculum*	*Cases of jaundice*	
			Number	*Per cent*
14	11	#38 pooled weekly serum specimens taken during pre-icteric period. Irradiated 45 min., 85% 2537 A. Dose 0.5 ml. 1 : 3 S.C.	0	0
15	13	Same serum used in Group 14, *non* irradiated. Dose 0.5 ml. 1 : 3 S.C.	2	15.4
16	15	Pooled dried plasma. One donor developed jaundice 4 days after bleeding. Dose 1 ml. S.C.	0	0
17	10	Serum from young man during spontaneously occurring jaundice. Dose 1 ml. 1 : 4 S.C.	4	40
18	3	Same serum used in Group 17. Dose 1 ml. 1 : 4 I.N.†	0	0
19	3	#38 pooled pre-jaundice phase serum. Dose 1 ml. 1 : 4 I.N.	0	0
20	20	#125 pooled pre-jaundice serum irradiated in thin quartz cell 2½ sec. by high energy low pressure water-cooled mercury vapor lamp. Dose 1 ml. 1 : 4 S.C.	0	0
21	9	Same serum as in Group 20 *non* irradiated. Dose 1 ml. 1 : 4 S.C.	1	11
Total	273		37	

* S.C.—Subcutaneously.

† I.N.—Intranasally.

could be shipped under suitable conditions. Many samples were taken and from these nine were selected for experimental use. These are tabulated in Table III.

EXPERIMENTAL STUDY

Volunteers were obtained in an institution with a population of about 1700. During the greater part of the study it was possible to use groups composed equally of both sexes. After 189 persons had been inoculated and 30 had developed jaundice it was found there was no apparent difference in susceptibility of the sexes and thereafter either sex available was used. Individuals with infectious diseases or history of recent antisyphilitic treatment were excluded. The age range was 13-57.

Table IV shows the size of groups used with brief descriptions of materials used for inoculation, and tabulation of cases of jaundice. Groups are arranged in chronological order in the table.

Each serum given was diluted with phosphate-buffered normal saline solution, pH 7.6, Berkefeld-N-filtered and cultured for sterility. Subcutaneous inoculation was always into the arm. Intranasal inoculation was done with the subject lying on the back of a table with the head dependent. Half the inoculum was dropped into each nostril and the subject then sniffed the material well back into the nose. Materials used for inoculation including sera and dried vaccines were stored routinely at 4° C. for variable periods up to a year. The maximum survival time of the icterogenic agent at this temperature was not determined.

All persons inoculated were bled just before inoculation and weekly afterward for 4 to 6 months. Bleeding was done on the same day of the week for each person and at about the same time of day, usually 3-5 hours after breakfast. The serum was separated the same day and a quantitative indirect Van den Bergh estimation was done.[25] Readings were made in a comparator using cobaltous sulfate standards.

Total leukocyte and differential counts were done weekly on the first few groups. No significant variation during the period of jaundice was seen in either count. Schilling counts were then done routinely each week and these failed to show any variation in jaundiced patients.

Moss blood grouping was done for each subject. No correlation between blood group and susceptibility to jaundice was found. Eighty-five sera were tested for Rh factor. Both Rh-positive and Rh-negative individuals were found susceptible to jaundice.

Some groups were skin tested just before inoculation with sera, one of which (Group 17) was icterogenic; 0.1 cc. of serum was given intracutaneously and the test was read 30 minutes later. Some slight reactions with small wheals up to 0.8 cm. in diameter with surrounding zones of erythema were seen. However, there was no correlation between the result of the skin test and susceptibility to jaundice, those subjects developing jaundice being equally divided, two showing small reactions and the other two no reaction. Non-icterogenic sera were also found to give similar slight reactions in some individuals.

The cephalin-cholesterol flocculation test of Hanger[26] was done weekly on each serum specimen, always with fresh serum on the day of bleeding. Invariably the test is strongly positive when clinical jaundice is present. In the subclinical range in our experience, the test may or may not be strongly positive. Many +, ++ and +++ reactions have been seen with sera from individuals who were in normal health so far

TABLE V

SUMMARY OF ALL EXPERIMENTAL GROUPS DEVELOPING JAUNDICE SHOWING INCIDENCE ACCORDING TO AGE GROUP

	Inoculated	*Developed jaundice*	*% developed jaundice*
10 – 14	2	0	0.0
15 – 19	35	3	8.6
20 – 24	45	10	22.2
25 – 29	25	4	16.0
30 – 34	27	8	29.6
35 – 39	17	4	23.5
40 – 44	13	3	23.0
45 – 49	9	3	33.3
50 – 54	2	2	100.0
55 – 59	1	0	0.0
Total.....	176	37	

as known. Difco antigen was used.

Icterogenic serum specimens have been repeatedly examined in the electron microscope by Lt. Don R. Mathieson of the Naval Research Medical Center. No particles of uniform morphology were found.

In Table V are listed all experimental groups in which cases of jaundice developed, according to age groups. The size of the groups is too small to give an accurate idea of the true susceptibility. The survey done in the Virgin Islands is undoubtedly much more reliable in this respect because of the larger number involved.

For statistical purposes jaundice was considered to be present when the serum bilirubin value was 1.0 mg. per cent or higher. Clinical jaundice was usually not seen until this value was 2.0 mg. per cent or more. In Table VI are listed all cases of jaundice occurring in experimental groups with data obtained from weekly blood specimens of each individual. In a few cases subjects were removed from the institution before complete disappearance of jaundice. A short statistical summary of 37 cases of jaundice is given in Table VII. Incubation period is here defined as elapsed time in weeks between inoculation and first appearance of a serum bilirubin of 1.0 mg. per cent or more. Duration of jaundice here

TABLE VI

OBSERVED DATA, CASES OF JAUNDICE ONLY

Group	*No.*	*Age*	*Incubation period, wks.**	*Duration of jaundice, wks.†*	*Time required for serum bilirubin to return to 0.5 mg. %, wks.*	*Highest serum bilirubin observed, mg. %*
1	2	17	14	1	3	5.0
1	7	15	13	2	3	3.0
1	8	24	16	2	3	1.1
1	12	22	14	1	2	1.0
1	13	33	13	3	4	10.0
1	23	46	16	3	3+	8.0
1	30	28	12	3	5	4.5
1	31	21	5	1	2	1.7
1	33	21	12	2	3	4.0
1	38	38	14	6+	6+	20.0
1	44	32	12	1	2	3.0
1	48	47	13	4	5	9.0
2	57	33	15	1	2	1.2
2	59	26	10	2	4	2.2
3	61	42	19	7+	7+	16.0
3	70	37	18	2	3	2.8
6	132	36	12	1	1	2.1
6	136	39	12	1	1	2.0
6	138	22	11	3	3	5.6
6	162	31	12	2	3	1.2
6	163	21	12	3	3+	2.6
6	167	24	10	2	3	4.8
7	150	33	8	2	4	5.6
7	176	21	10	1	2	2.8
7	179	40	10	4	6+	2.6
8	188	32	17	2	3	4.8
10	201	24	13	2	2	2.4
10	208	19	8	1	2	3.2
10	217	26	9	2	4	2.4
10	220	22	10	3	4	8.0
15	112	28	14	3	4	3.3
15	125	43	9	3	4	5.4
17	266	31	20	1	1	2.1
17	271	51	19	3	4	6.2
17	272	46	16	4	5	10.0
17	273	52	20	1	2	2.1
21	306	30	10	2	4	2.4

* Elapsed time until serum bilirubin value of 1.0 mg. % or higher is found.
† Time during which serum bilirubin of 1.0 mg. % or more is found.

TABLE VII

STATISTICAL SUMMARY OF CASES OF JAUNDICE

	Range	*Average*	*Median*	*Mode*
Incubation, period, weeks	5 – 20	12.9	12	12
Serum bilirubin, mg. % (maximum observed)	1.0 – 20.0	4.7	3.0	
Duration of jaundice, weeks*	1 – 7	2.35	2	2
Serum bilirubin elevated, weeks†	1 – 7	3.3	3	3

* Number of weeks during which serum bilirubin value was 1.0 mg. % or higher.
† Number of weeks required for serum bilirubin to return to 0.5 mg. % or less.

means the time in weeks during which the serum bilirubin remained at 1.0 mg. per cent or higher.

In two cases of jaundice a biphasic rise in serum bilirubin was seen. Both cases occurred in group 1. In one case the first rise occurred after 3 weeks and reached 1.1 mg. per cent. In the thirteenth week a second rise to 3.2 mg. occurred with a further rise to 10.0 mg. the following week. In the other case the first rise occurred after 5 weeks reaching 1.8 mg. in the eighth week with a secondary rise to 1.7 mg. in the fourteenth week.

Clinically most cases were quite mild; epigastric discomfort and nausea were commonly present shortly before jaundice appeared. Vomiting occurred in a few cases. Clay-colored stools and dark urine were present during jaundice. Slight fever up to 100°-101° F. was noted in a few cases at about the time of onset. Anorexia was usually present during jaundice. Most jaundiced patients remained ambulatory. Dermatitis and arthritis were not seen.

So far as known no contact cases of jaundice occurred. Only 3 cases have appeared among 1,400 uninoculated individuals not included in this study during the past 18 months in the institution's population of about 1,700. One of these spontaneous cases was found to have carcinoma of the liver. During one period of 4 months 40 uninoculated persons having close contact with the inoculated group in which jaundice was occurring were bled weekly and no evidence of jaundice was found among them.

Colonel Stanhope Bayne-Jones supplied sera from three persons who

had acted as donors to pools of serum used in preparation of batches of yellow fever vaccine which had produced jaundice in the United States Army during 1942. It was subsequently found that all three of these persons had histories of jaundice occurring several months before they were bled for the vaccine. Two of these sera were used for inoculation in groups 12 and 13. No cases of jaundice appeared in these groups.

The plasma used in group 16 was a part of one lot prepared commercially. Before the lot was released it was learned that one of the donors to the pool had developed jaundice a few days after being bled. The whole lot was immediately confiscated. On July 22, 1943 a group of 15 were inoculated with 1 cc. each of this material subcutaneously. No cases of jaundice developed. On December 10, 1943, another group of 10 were inoculated with the same material in a dose of 10 cc. intravenously. To date no jaundice has appeared in the second group.

The serum used for inoculation in group 17 was obtained from a boy 18 years old, an inmate of the institution, who developed jaundice spontaneously. He had had no known contact with members of the experimentally inoculated groups. Serum was obtained from him five days after jaundice had appeared, at which time he complained only of poor appetite; there was no fever, the serum bilirubin value was 12 mg. per cent; the Hanger reaction was strongly positive. This case and the 4 cases which subsequently developed in those inoculated from it were indistinguishable in appearance from the other cases seen. The incubation periods ranged from 16 to 20 weeks.

Reports of two cases of jaundice among the personnel of one Coast Guard vessel were received in 1943. Both men were vaccinated the same day with one lot of "aqueous base" yellow fever vaccine containing no serum. One developed jaundice in forty-nine days, the other in 129 days. The findings in both cases, as reported, were consistent with the finding in cases of our series. On November 11, 1943, thirteen subjects were inoculated with twice the recommended dose of the same lot of vaccine mentioned above as used on the Coast Guard vessel. To date no cases of jaundice have developed in this group.

Attempts were made to transmit jaundice to animals, using materials which had produced the disease in humans. These included yellow fever vaccine as well as sera derived from patients in the Virgin Islands and from persons with experimentally produced jaundice. Monkeys, pigs,

rabbits, guinea pigs, white rats, Swiss mice, cotton rats and Syrian hamsters were employed. In no instance were we able to produce any illness in experimental animals which could not be accounted for by other agents and no animals became jaundiced as determined by physical or chemical examinations.

E. W. Goodpasture supplied specimens of liver from fatal cases of jaundice which occurred following the use of yellow fever vaccines. Extracts of these specimens were made both with 95 per cent alcohol and normal saline solution. The extracts were used as antigens in complement fixation tests designed to determine whether or not serum antibodies were present following this type of hepatitis. In a limited series of tests negative results were obtained. Attempts were also made to produce a complement-fixing antigen in the developing chick embryo; these were inoculated both in the allantoic sac and the yolk sac. Embryos 9-10 days old were inoculated with icterogenic yellow fever vaccine. Serial transfers were made in both series at intervals of 3-4 days and at 1 week. No fixation resulted with allantoic fluid or yolk sac emulsions tested against human sera obtained after recovery from jaundice.

Comment

It was recognized in 1942 during an epidemic of jaundice in the United States Army that some agent in human serum employed as a diluent in yellow fever vaccine was probably responsible. The yellow fever vaccine now in use does not contain serum. The two cases described above of jaundice in personnel of the Coast Guard following administration of serum-free vaccine may well have been due to other causes than the vaccine and probably should be so regarded unless evidence to the contrary is obtained.

It is difficult to evaluate the claim of Findlay and Martin[16] to have produced jaundice in 3 of 4 subjects inoculated with nasal washings of patients in the early stages of post-yellow fever vaccine jaundice. Two of these cases are claimed to have been subclinical jaundice. In one, T.P., the icteric index did not exceed 8 units. In the other, M.M., the only direct evidence of jaundice was a weakly positive direct reaction (serum Van den Bergh). So far as known no cases occurred among familial contacts of thousands of cases of jaundice occurring in the U. S. Army and it is felt that additional evidence should be obtained before concluding that the condition may be transmitted by the respiratory tract.

Since jaundice has repeatedly followed the administration of whole blood or blood products there is an urgent need for some means of detecting the presence of the jaundice-producing agent in the blood, or for some practical method for treating blood products so that the danger of jaundice following their use may be eliminated.

Summary

Results of a sample survey of an epidemic of jaundice occurring subsequent to vaccination against yellow fever in the Virgin Islands in 1942 are given.

Jaundice was produced experimentally: 1. By the inoculation of two lots of yellow fever vaccine containing human serum. 2. By the inoculation of small amounts of filtered serum from each of three individuals and of a serum pool from nine individuals all of whom had previously received yellow fever vaccine containing human serum. 3. By inoculation of serum from one individual who had early spontaneously occurring jaundice.

Two sera which were icterogenic when inoculated subcutaneously failed to produce jaundice by the intranasal route.

Both sexes are apparently equally susceptible to this type of jaundice. Those of all four Moss blood groups and both Rh-positive and Rh-negative persons were found to be susceptible.

Susceptible persons did not give uniform local skin reactions to icterogenic sera.

The jaundice-producing agent is filterable, survives drying in vacuum, storage for long periods in serum at 4° C. and heating to 56° C. for one-half hour in the dried state. The agent was found to be present in the blood during the pre-jaundice period but not 2½ months after the disappearance of jaundice.

The icterogenic agent is apparently inactivated by short exposure to ultraviolet irradiation.

Transmission of jaundice by ordinary contact apparently did not occur during this experiment.

Attempts to produce jaundice in experimental animals were unsuccessful.

Antigens prepared from human livers and from chick embryos failed to fix complement in the presence of sera obtained after recovery from jaundice.

Attempts to visualize virus particles in icterogenic sera by electron microscopy were unsuccessful.

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MAJOR THERAPEUTIC TRENDS IN AMERICAN PSYCHIATRY*

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IN THIS year of titanic struggle, the attention and effort of all are engaged in the greatest argument which has ever agitated the human race. The central issue in dispute is concerned fundamentally with forms of social organization, and differing attitudes as to what is humanly tolerable in the coercion of one group by another. The overreaching aggression of certain nations has ultimately mobilized against them the united sentiments and the massed forces of the rest of the world in bitter combat, bloodshed and devastation. In a world aflame with passionate strife, a small handful of us are gathered here this evening to consider together briefly a certain portion of the healing art, psychiatry, and to scan in a quick review the changes wrought in this art, and in its fundamental scientific concepts, by a century of effort on the part of our American predecessors and contemporaries. My assignment in this program is to discuss the major therapeutic trends.

Surely, it would seem, there could be no greater contrast than that between the most bitter war in history and the gentleness of a healing art devoted to the relief of the distraught mind. A decent respect for historical truth compels me to admit, however, that the record of this gentle art has been often punctuated by disputes of amazing bitterness and rancor. This unpleasant fact was brought to my mind with special force a few years ago, at a psychiatric convention. I met by chance in the lobby at the same moment two friends of mine, both eminent and successful psychiatrists, and one of them refused to shake hands or to speak with the other because of an enmity which had arisen over a difference of belief as to psychotherapeutic principles. Differing attitudes regarding proper principles of exercising therapeutic influence have a great power to arouse antagonism. This fact has had

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a crucial influence in determining therapeutic trends in psychiatry.

In the early decades of the 19th century when American psychiatry began, it took on social form and institutional substance as a humanitarian movement, motivated by kind sentiments, as expressed, for example, in the terms "Retreat" and "Asylum." The leading idea was not primarily that of therapy, but of care; nevertheless the responsibilities were entrusted to physicians and this fact made it inevitable that therapeutic impulses would have a large influence. Indeed Pinel's courage and success in trying out "moral management" had set a brilliant and encouraging therapeutic challenge in closest alignment with the humanitarian motives.

In my judgment the most useful brief review of major therapeutic trends in American psychiatry should concern itself primarily with psychotherapeutic orientations. In making the choice to emphasize the psychotherapeutic side of psychiatric work I am aware that one invites some of the unpleasant emotional reactions which seem always to have troubled the earnest discussion of this topic, yet I deliberately make this choice because it seems to me that the study of the psychotherapeutic trends provides the most penetrating glimpses into the general philosophy and therapeutic aims of the different psychiatric workers and different periods.

As the introductory scene in this review let us consider a few events in the two year period 1875 and 76, not because that was the historical beginning, but because certain dramatic events of that period give a useful cue to a fundamental theme for this brief essay.

A story is told regarding George M. Beard's paper before the American Neurological Association at its second annual meeting in 1876. Beard discussed the power of suggestion in the production and relief of symptoms. In the heated avalanche of contrary opinion which followed, William A. Hammond, who has been called the father of American neurology, is said to have stated angrily that if Beard's doctrine of psychological influence were accepted he would feel like throwing away his medical diploma and joining the theologians! This contemptuous rejection probably expressed quite well the attitude of the medical profession at that time toward psychotherapeutic methods.

This incident was approximately contemporaneous with the publication (in 1875) of the first edition of Mary Baker Eddy's *Science and Health*, to be followed in a few years by the appearance of the *Journal*

of Christian Science. It is interesting to note that Pierre Janet, in his extensive treatise on *Psychological Healing* gave greater emphasis and more space to Mary Baker Eddy than to any other American.

While acknowledging that the degree of institutional success of the Christian Science movement was phenomenal, the urge toward the development of its doctrines is not difficult to appreciate. Clinical experience shows us repeatedly how the recovered patient tends to overgeneralize the principles which have helped him regain health. Thus, Mary Baker Eddy, after the cure of her hysterical paralysis by Phineas Quimby, formulated her therapeutic gospel in a religious form since that was the only extensive ideology available to her for large-scale philosophizing. The phenomenal growth of the Christian Science movement as a religion constituted an indirect indication, however, of the culpable neglect of psychotherapy by the medical men of that period; and then, in turn, the overdrawn tenets of the Christian Scientists and the disastrous neglect of elementary medical considerations by the healers of that cult certainly antagonized the medical profession and made even more difficult the sane and rational development of psychotherapeutic study as a part of the medical art.

In 1875, the year before Beard's paper, we may note the first exposition of Weir Mitchell's "rest cure," in Seguin's Series of American Clinical Lectures. Weir Mitchell's "rest cure" would now be considered by all as having been essentially a psychotherapeutic method, whose efficacy depended more on its unavowed psychological features than on its direct effects on "Fat and Blood," as Mitchell himself claimed. It seems probable to me that this excessively somatic mode of rationalizing his therapy reflected also the prevailing antagonism against psychological healing, just as did Beard's experience, but in the opposite sense. That is to say, Mitchell, being emotionally well attuned to the prevailing attitudes of medical leadership, was inclined automatically to steer away from the psychotherapeutic principles, whose borders he was skirting, and to formulate his undoubtedly successful experiences in the more acceptable somatic ideology. Mitchell may have understood that in addressing his medical colleagues he had to express himself in an idiom acceptable to medical prejudices. Here is a consideration worth the careful attention of anyone who would seek to understand the psychiatric practices of men of another generation. We have grown accustomed in our day, at least some of us have grown accustomed,

to speak of our psychotherapeutic efforts and experiments in fairly forthright fashion, not deeming it so necessary as formerly to neurologize our thinking, nor to hematologize, nor to vascularize nor otherwise to try to translate into some other form of medical jargon the human facts with which we deal. When reading any old writer on the medical arts we should, I presume, prepare and utilize for each a sort of conceptual slide-rule which would help us to calculate back into actual behavior the actions and words which he may have used, but which, for publication, he had laboriously elaborated into the special medically acceptable idiom of his time and of his personal predilection.

To be more specific, let us consider Benjamin Rush. He is spoken of as the father of American psychiatry. His portrait is the emblem of the American Psychiatric Association. He developed the first systematic theory of insanity formally propounded in America. He considered insanity to be a disease of the blood vessels, and his enthusiasm for blood-letting as a cure for madness was widely recognized here and abroad. I quote from the 1827 edition of his *Medical Inquiries and Observations upon the Diseases of the Mind*. Rush wrote: (p. 15) "Having rejected the abdominal viscera, the nerves and the mind as the primary seats of madness, I shall now deliver an opinion, which I have long believed and taught in my lectures, and that is, that the cause of madness is seated primarily in the blood-vessels of the brain, and that it depends upon the same kind of morbid and irregular actions that constitutes (sic) other arterial diseases."

Having thus clearly formulated in organic terms his conception of the *proximate* cause of madness, Rush proceeded to a consideration of the *remote* and *exciting* causes. I quote again (pp. 44-45): Madness "is more common from mental than corporeal causes: . . . I have taken pains to ascertain the proportion of mental and corporeal causes which have operated in producing madness in the Pennsylvania Hospital. . . . In 50 (patients), 34 were from mental causes and 16 from corporeal causes . . . but I am sorry to add, my success in this inquiry was less satisfactory than I wished. Its causes were concealed in some cases and forgotten in others. . . . (p. 44) Depression of mind may be produced by (events) that are forgotten; or by the presence of objects which revive the sensation of distress with which it was at one time associated, but without reviving the cause of it in the memory." (This last sentence could be elaborated into an interesting premonition as to the importance

of unconscious mentation.)

Benjamin Rush then gave considerable attention to the methods of therapy which are intended to act through the medium of the mind. Speaking particularly of hypochondriasis, which he called a partial intellectual derangement or partial madness, Rush wrote (p. 104): "The first thing to be done by a physician under this head, is to treat the disease in a serious manner. . . . However erroneous a patient's opinion of his case may be, his disease is a *real* one. It will be necessary, therefore, for a physician to listen with attention to his tedious . . . details of its symptoms and causes." (Here is a shrewd observation of an important psychotherapeutic consideration.)

Rush also advocated employment or business of some kind as a psychotherapeutic measure. He quoted Burton with approval, "Be not idle, be not solitary," but he capped it with a more brilliant suggestion from Johnson, "When you are idle, be not solitary; when you are solitary, be not idle."

Rush has noted, also, (p. 124) the psychotherapeutic device, in dealing with depressed patients, of "mentioning the *name* of a parent, relative or friend" or "carrying the patient's memory or imagination back to the innocent and delightful sports and studies of early life."

These statements by Rush concerning the mental causes and the mental treatment of "madness," are quoted in order to illustrate his knowledge of psychopathological and psychotherapeutic truths, and also to raise the question as to how he reconciled them with his vascular theory of the cause of madness.

I have not found that he offered a fully systematic exposition as to how the mental causes and mental therapies operated upon the *presumed* arterial disease, but a significant indication is given in his discussion of the *indirect causes*, (p. 36): "Madness is excited in the understanding most frequently by impressions that act primarily upon the heart. . . . Some of these . . . are joy, terror, love, fear, grief, distress, shame, . . . inordinate love of praise, domestic tyranny, and the complete gratification of every wish of the heart."

This quotation offers the needed cue, I believe, to the manner of thinking by which Rush was able to combine his psychopathology and psychotherapy of insanity with his vascular theory of etiology. The heart and blood vessels were conceived of by him as the instrumentalities of emotional experience—the physiological mechanism through

which transient influences of an emotional kind produce more enduring effects. His vascular theory of etiology was thus, in one sense, an emotional theory, conceptually somaticized, so to speak, in somewhat the same fashion that Kempf has utilized the autonomic nervous system for the conceptualization of the nature of emotional attitudes.

This brief excursion into the writings of Benjamin Rush is undertaken with the most sympathetic appreciation of his keen observations and shrewd practical sense, yet I wish also to point out, through this illustrious illustration the great defect which I think has limited and hindered Rush, and Weir Mitchell, and others in their attempts to formulate psychotherapeutic principles. This defect is the lack of *psychological depth* in their professional formulations. Ideas about the heart and arteries, or, in Mitchell's case, ideas about fat and blood, have afforded a gratifying basis of rapport with their professional brethren, and served to save them from any threat of professional excommunication, but an unfortunate result was to maintain a level of comprehension too superficial to support the formulation of sound psychotherapeutic *strategy*. Psychotherapeutic tactics were offered in abundance, but not strategy. Consider, for example, this recommendation by Rush for the treatment of a certain specific hypochondriacal complaint (p. 108): "Cures of patients, who suppose themselves to be glass, may easily be performed by pulling a chair, upon which they are about to sit, from under them, and afterwards showing them a large collection of pieces of glass as the fragments of their bodies." Although this manoeuver may perhaps have had some efficacy as a tactical device for defeating a specific hypochondriacal delusion, we would certainly not, in our day, recommend it as a well-considered part of psychiatric strategy in helping such a patient toward sound and enduring health.

The deficiency in psychological depth, and the consequent preoccupation with psychological tactics to the relative neglect of psychological strategy, also tended to vitiate in considerable degree the case-studies on which further progress might depend. Based on a vascular theory of mental derangement the pertinent medical history, as also the course of the disease under treatment, was conceived of as a history of the individual's arterial system. The importance of emotional events was appreciated, but primarily, it would seem, because they affected the heart and thereby the arteries of the brain. Instead of seeking to

understand the biographical development of a personality, as a basis of psychotherapy, what one sought was a catalogue of incidents which might be considered to have affected the arteries.

In the middle of the last century the German psychiatrist Wilhelm Griesinger had a considerable influence in America, as elsewhere, in shaping conceptions of psychiatric therapy.

Now Griesinger is customarily considered to have been the great proponent of the somatological as opposed to the psychological concept of mental disorder. It is true that he was an ardent, and very effective, champion of the idea that insanity is a cerebral disease, and he took great pains in seeking to relate the knowledge of mental disorders current in his day to whatever conditions of the cerebral tissues he could establish or reasonably conjecture; yet he too, as well as Rush, indicated clearly his belief that psychic causes were the most frequent and the most fertile causes of mental disorders. It seems reasonable to assume that many American physicians, reading his textbook may have read through to the back of the book and been influenced in their practical work by his very sensible discussions there of psychotherapeutic measures. I quote several passages from Robertson and Rutherford's translation of the second edition of Griesinger's textbook *Mental Pathology and Therapeutics,* (p. 327):

"Theoretical hypotheses have rendered it difficult for science to recognize the results of experience. . . . It must be remembered that cerebral activity may be modified quite as effectually, directly and immediately by the evocation of frames of mind, emotions, and thoughts, as by diminishing the quantity of blood within the cranium, or by modifying the nutrition of the brain. . . . We have, in the direct provocation of certain states of mind, a very powerful means of successfully modifying disturbances of the somatic state." (p. 328) "Nowhere is it of greater importance than in the treatment of insanity, to keep in view the individual; nowhere is the constant consciousness more necessary that it is not a disease but an individual patient that is the object of our treatment. . . . A penetration into the psychical individuality of the patient is here demanded, which is scarcely ever necessary in ordinary medical practice." . . . (p. 343) "The old *ego* which in insanity for a long time is not lost, but only superficially repressed, or hidden in a storm of emotion, behind which it remains for a long time capable and ready to re-establish itself, must, as far as possible, be re-

called and strengthened. . . . The moral treatment of insanity is most successful when the *ego,* already formed, fixed, and only temporarily repressed . . . waits the opportunity to resume its former place." (p. 90) "The inquiry into the history of the case ought to embrace the whole of the bodily and mental antecedents of the individual. . . . We must faithfully and intelligently comprehend the relation of the predispositions . . . the education and the governing inclinations of the individual. . . . Only in this way is an insight into the true history of these diseases possible; only thus can we succeed in grasping at their beginnings those fine threads which have ultimately entwined themselves into delirious conceptions; only thus can we . . . recognize the far-back commencement of the preparation for the illness. . . . All of this is of the highest importance in a system of treatment which gathers from the history of the case indications, sometimes for the amelioration of inveterate chronic processes, at other times for the removal of certain psychic causes, and which requires a profound knowledge of the character of the individual to enable us to employ all his inherent resources in support of our active treatment."*

These quotations from Griesinger provide an admirable psychotherapeutic orientation, yet I presume they pictured an ideal, rather than an actual trend in practice. Whoever may have attempted to put this ideal into practice must have been greatly handicapped by the lack of specific guidance. Griesinger presented a much more sophisticated and comprehensive view than did Rush, of what psychiatric therapy might include, but here again the preoccupation with states of the cerebral tissues probably served to hinder the more thorough study, which might otherwise have been carried through—and perhaps brilliantly carried through—regarding the organization and growth of personality and the possible strategic utilization of principles so derived in a more explicit and constructive type of psychiatric therapy.

Throughout the nineteenth century American psychiatry was affected from time to time by reverberations of the European interest in hypnosis as a psychotherapeutic procedure, but, in general, hypnosis fluctuated about the charlatan fringe until near the end of the century, when the Nancy school attracted some American medical men, and Boris Sidis and Morton Prince made extensive use of hypnosis. The

* These statements do not differ essentially from those in the first German (1845) edition, except the quotation from p. 90 which represents a part of the revision for the second edition.

work of Sidis never got into the main stream of psychiatric development, and even Morton Prince felt somewhat outside the main current. When he wished near the end of his life to set up at Harvard the means for continuing researches in medical psychology, it was not done through the medical school or associated hospitals but through the college in Cambridge.

These nineteenth-century trends in psychotherapeutic thought in America to which I have been referring, illustrate, I think, a curious result of the emotional strains aroused among medical men in the consideration of psychotherapy. The Christian Science movement bore indirect witness to this aversion, in taking some principles which had genuine psychotherapeutic significance and expanding them extravagantly into a religious dogmatism, sharply delineated from the medical profession, and even belligerently opposed to medicine. The unpleasant experiences of George M. Beard and of Morton Prince when identifying themselves frankly with psychological modes of treatment were clearer indications of this aversion. But perhaps the more pernicious effects of this medical attitude were exhibited in the work of Benjamin Rush and Weir Mitchell. Steering clear of this aversion, Mitchell cast his psychotherapeutic work into the pattern of a nutritional theory, and Rush put his in terms of arterial disease, both of them failing thereby to establish any psychological depth for their keen psychological perceptions.

The twentieth century began more propitiously for the medical development and use of psychotherapy. In the first decade the invitations to two distinguished Continental visitors, Pierre Janet and Sigmund Freud, bore witness to a considerable desire in this country for psychological insight. Also, in ways indirectly affecting the medical world, the pragmatism of William James and the integrative functionalism preached by John Dewey began to modify academic psychology in directions more readily assimilable to the practical tasks of the physician. Adolf Meyer as a pragmatic clinical scholar, with talents and achievements which commanded medical respect, championed and established a clinical viewpoint, pluralistic yet integrative, in which symbolic intellectual functions and diffuse regulative emotional functions of the organism were simply and regularly required to be taken into the medical account. This view met with somewhat less negativistic resistance than had been felt against presumed "psychological mys-

teries." The one article by Dr. Meyer which has seemed to me to epitomize most aptly his fundamental influence on psychotherapy was the one published in 1915, under the characteristic title "Objective Psychology or Psychobiology with Subordination of the Medically Useless Contrast of Mental and Physical." By the time of its publication, Meyer's influence had already been widely effective in this direction.

I quote one sentence from that paper: "As soon as mental attitudes and mental activities are accepted as definite functions of a living organism, mentation and behavior is treated as a real chapter of the natural history of man and animal, and psychology ceases to be a puzzle supposedly resisting the objective methods of science."

In my studies of Meyer's papers, I cannot see that the development of his very comprehensive views hinged on any one crucial point, but one can see a very important part of his thought in his early conception of the dementia praecox type of reaction as determined in part by bad mental habits, offering therefore the possibility of change.

Meyer did not systematically elaborate specific techniques of psychotherapy. Even the well known Meyerian expression "distributive analysis and synthesis," which was systematically expounded by his pupil, Oskar Diethelm, represented a considerable eclecticism, and Meyer has preferred to consider psychiatric therapy as the application of common-sense to special individuals and situations in the light of all the available facts.

It is no secret that Meyer deplored the denominational features of psychoanalysis; yet, in a paradoxical way, I am inclined to think that the measure of good will which psychoanalysis has gained in the medical profession in this country, and the decidedly medical affiliation and orientation of psychoanalysts here as compared to the Continent, has depended in considerable measure upon the influence of Meyer, and his broad psychobiological orientation in medicine. Certainly the psychotherapeutic trends which Meyer cultivated were strongly opposed to superficial "neurologizing" and strongly encouraged a truly deep and genuine psychological insight.

Since Meyer's breadth of view and depth of understanding brought about in his practice and teaching a medically acceptable meeting ground, the organicist shibboleth has lost much of its divisive force in American discussions of therapy.

William Alanson White also exerted an extraordinarily powerful influence on the psychotherapeutic orientation of American psychiatry. It is of special interest to consider the manner in which he dealt with the organicist medical tradition. In a paper published just 50 years ago, he discussed in a broad way the lesions of the brain which cause cerebral malfunctioning. It is clear in this paper that White's way of thinking at that time required that some lesion be postulated as the cause of any cerebral malfunctioning. He expounded in this paper the concept of two orders of lesions, "molar" and "molecular." Having achieved in theory a molecular dispersion of the rationalistically-required lesion, he subsequently felt free to deal with the personal functioning of the organism, in whatever practical ways might be found, without the compulsion to hypothecate lesions. He had a special talent for administrative leadership and a warm enthusiasm for what could be found practically workable in the Freudian contribution, or in any other promising line of thought; and this combination did much to shape a therapeutically oriented American viewpoint in psychiatry.

The development of psychoanalytic therapy has had a profound influence on all psychotherapeutic work in America. In a direct way, we have had the influence of Dr. Brill, his own work and his translations of Freud's work, and we have had a considerable influx of European analysts and a very extensive training of American analysts. It would be difficult to disentangle which might be characteristically American from the rest of the psychoanalytic developments here. Aside from the technical details of free-association or dream-analysis, and rather independently of psychoanalytic doctrines, psychoanalytic therapy has developed in its practitioners and in those who have been privileged to keep in touch with such work, a solid working-appreciation of personality depth, which has in one way or another, influenced all recent psychiatric thought.

It does not seem profitable at this time to attempt a very detailed consideration of the manner in which specific psychoanalytic discoveries and doctrines have influenced therapeutic trends. It would be very easy to get caught up in minor acrimonious disputes. Yet there is one point concerning psychoanalytic history which affected the general trend of psychiatric therapy in so significant a degree, in my opinion, that I should not feel it proper to pass it by without at least the expression of a personal opinion.

It seems to me that the third decade of the psychoanalytic movement, approximately the time of the first World War, marked a special crisis. What was dynamic and vital in the psychoanalytic appreciation of personality depth had come perilously near to being suffocated in a verbalistic game of erotic symbols and libidinal formulations; but the stirring events of that period and the psychiatric casualties of war brought insistently to attention the need for a broader understanding of *anxiety*, as a fundamental problem in human adjustment, and of *aggression* as a factor in a personal maladjustment. For many psychiatrists also the war brought a more concrete meaning to the concept of the self-defensive nature of neurotic reactions. Not only did the psychoanalysts, as a special group, utilize these considerations for broadening their concepts; other psychiatrists came thereby to a better appreciation of the pathological forces and mechanisms with which they had to cope, in their psychotherapeutic endeavors.

At this point in the discussion it is appropriate to consider a fundamental social principle of modern psychiatric work. I refer to the increased appreciation that neurotic and psychotic reactions, considered as poor or second-rate types of adjustment to life-situations, arise as responses to the tensions of interpersonal attitudes. Somehow, without there being any specific date or situation to which we can allocate the discovery, interpersonal relationships, or rather interpersonal attitudes, have become the very special business of the psychotherapist. Under the intellectual leadership of Harry Stack Sullivan, "Interpersonal relations" have indeed become the subject matter of the special journal which is entitled "Psychiatry." It is a primary aim in the strategy of psychiatric therapy, now, to help the patient to gain emotional security and self-assurance in interpersonal relations. This fundamental trend has many ramifications in current psychiatric practice and research, of which one example requires special mention: the widespread experimentation in group therapy, in which the interactions between the members of a group are utilized for therapeutic purposes.

The rather sudden and remarkable development of child psychiatry in America has been occurring at a time when psychiatrists have been specially preoccupied with concepts of personality structure and personality growth. The increasing appreciation that the childhood years have great strategic importance for subsequent mental health or disorder, stimulated efforts to help children already beginning to exhibit

behavior-problems, and conversely experience with children has served to generate optimistic faith in the potentialities of personality growth, with a bit of help, even under many hindering circumstances. Frederick Allen, in his recent book "Psychotherapy with Children" has given a specially earnest and appealing presentation of a very important modern trend in psychotherapeutic philosophy which rests on a belief in the dignity of human nature and a respect for individual difference. Translated into methodology, such a psychotherapy avoids any dominating attitude, yet avoids inert passivity, and seeks to favor such respectful interaction between physician and patient as encourages the spontaneous and self-assured assumption of a responsible and gratifying social role.

In tracing hurriedly certain main therapeutic trends, I have given primary attention to psychotherapeutic considerations, chiefly because they have served to reflect the general working progress, and incentives, in psychiatry. Even the outspoken organicists whom I have quoted have indicated that in their experience, mental forces seem the main consideration. There is also a further reason. Psychotherapeutic experience and thought have largely shaped the growing concepts of *personality*, which at present dominate psychiatry to the extent that the term "personality disorder" is often now used as a practical synonym for any condition which is thought to need psychiatric attention.

Many of us are customarily disposed, now, to look upon the organic and toxic factors in psychiatric conditions as influences which *limit* or *hinder* the person in his adjustive behavior, spoiling his good performance and providing the occasion thereby for poor personal functioning in a manner largely determined by the personality. This formulation represents in my opinion, a more useful and stimulating view, than to look to the organic conditions as the direct causes of specific items of pathological behavior. It is nevertheless considered exceedingly important, for the purposes of psychiatric therapy, to combat these noxious organic or toxic conditions with the best means available, and to seek better methods of doing so; also to enlarge our knowledge of these noxious organic influences. Endocrinological dysfunctions and nutritional deficiencies also receive attention, by a similar rationale, in psychiatric treatment.

The impressive success achieved during our generation in the treatment of syphilitic disease of the brain has been an enormous stimulus

to the therapeutic enthusiasm of psychiatrists. The success of Wagner-Jauregg's malaria treatment of general paresis undoubtedly served to justify and encourage therapeutic trials of very bold measures for other psychiatric conditions, culminating in the shock therapies. The Sakel method, utilizing hypoglycemic coma induced by insulin, for the treatment of schizophrenic conditions, was the starting point about ten years ago for an extraordinary trend in psychiatric therapy, which may be characterized as the employment of a direct and violent attack upon cerebral tissues considered to be functioning in a pernicious way. This trend is still strong. It has brought us prefrontal lobotomy and metrazol, also. The favorite technique in shock therapy now—the use of the faradic electrical current applied to the skull—has greatly changed the current practices in the treatment of depressive states, by putting into the hands of the psychiatrist a means, often successful, for interrupting quickly the guilt-laden ruminations of the patient. The throwing of the electric-switch has not solved all the personality problems of these patients, but this method has added a new and powerful tactical resource, whose possibilities challenge us to learn how to employ it in a sound psychiatric strategy.

SHOULDER PAIN AND DISABILITY*

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AT first glance one would think of this subject as one of rather limited scope. Immediately, however, on investigation of one's clinical experience with pathology and symptomatology in this region of the body, one's respect for the multiplicity of abnormal conditions which may here cause disability or symptomatology increases. Respect is enhanced and interest intrigued by the structural and mechanical difficulties arising in this location tending to prevent improvement of function or relief of symptomatology when abnormal conditions are present. Even should one limit one's self to a consideration of just the shoulder joint and not the shoulder region, a great amount of material would present itself for discussion. This afternoon we shall consider the shoulder in its broader sense. If, therefore, we discuss briefly some phases of pathology and symptomatology and slight others altogether, we hope for your indulgence in order that we may refer to certain lesions in greater detail which we consider more common or more important.

One can group all pathology causing pain or disability in this region under six convenient headings:

1. Distant lesions causing radicular pain
2. Pathology of the muscular structures
3. Lesions of the acromio-clavicular joint
4. Arthritic pathology
5. Pathology in the bony structures
6. Lesions giving symptomatology due to or simulating that of tendon or bursal pathology

1. *Distant lesions causing radicular pain*

Pain located in the shoulder region, and frequently sharply circumscribed to the shoulder joint region, arising from pathology at a distance, is well known to occur. The most common cause of such discom-

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fort is probably radicular pain caused by irritation of nerve roots as a result of pathology in the cervical spine. The formation of arthritic spurs encroaching on the intervertebral foramen or the lessening of the diameter of these foramina by loss of disc substance due either to arthritic change or trauma frequently causes such manifestations. Small subluxations or unreduced dislocations are frequently first noted due to radiation of pain to the shoulder. Cervical rib or the scalene syndrome, while classically distributing pain and other symptomatology farther along the upper extremity, frequently early in their course cause radiation of pain to the shoulder. Syringomyelia in the cervical region may produce as one of its major symptoms a Charcot destruction of the shoulder joint. The above facts are so well known to this audience that no apology need be made for stating that cervical x-rays are necessarily a part of any satisfactory examination of the shoulder region where difficulty arises in diagnosis. Not only does interference with the direct nerve pathways to the shoulder cause pain here, but it is interesting to remember that pathology as far away as the gall bladder on the right or cardiac lesions on the left may be responsible for symptomatology in this location.

Fortunately, difficulty in ruling out true shoulder pathology in all the above lesions is slight. This is the only one of our group in which, on careful examination, no pathology is found in the shoulder itself. There is no loss of motion, spasm, protection, localized tenderness, deformity, paralysis, displacement of bony structure, change from the normal contour, or pathological x-ray findings. This does not mean that location of the pathology causing referred pain is in itself not difficult. It well may be. Absence of the above findings, however, immediately limits one's search and refers one's attention elsewhere.

2. *Pathology of the muscular structures*

Because of the complexity of the muscular arrangement and the fact that stability of this joint is mainly provided for by muscles, simple myositis as a result of overuse is frequently encountered. In addition to this, lesions causing general muscle irritation, such as trichinosis, may present continuing symptomatology here long after it has disappeared elsewhere in the body. We have encountered two cases of trichinosis simulating disability such as would ensue from injury to the capsule or the short rotator tendons of the shoulder. A relatively rare but extremely interesting condition, myositis ossificans, frequently begins in this location.

Paralytic lesions in this region are likewise of more prolonged duration and more difficult in treatment because of the mechanical disadvantages present. Poliomyelitic manifestations especially of the deltoid demand prolonged support in an awkward position which is frequently difficult to enforce. Paralysis of the serratus magnus muscle causing winged scapula not only demands prolonged support but such support is difficult to obtain. Axillary nerve damage is an extremely difficult lesion to treat. This nerve is almost impossible to repair if torn and the lesion is frequently overlooked for a considerable period of time.

Fortunately, in spite of the difficulties presented, much may be done for improvement and alleviation of dysfunction even in the more serious type of muscular lesion. This is well shown in cases of Erb's palsy where release of tight muscular structures may massively overcome the deformity present as well as in paralysis where transplantation of muscles may restore at least partial function.

3. *Lesions of the acromio-clavicular joint*

Lesions of this joint, being structurally near the shoulder joint itself, frequently are confused as being the cause of symptomatology in the shoulder. Careful and critical examination will quickly establish the true location of pathology. In spite of this we frequently see mis-diagnosis due to dislocations of the acromio-clavicular joint. Probably even more frequently we find infectious arthritis of the acromio-clavicular joint (especially tuberculous arthritis) mis-diagnosed as shoulder pathology.

4. *Arthritic pathology*

Probably more cases of shoulder dysfunction belong truly under this classification than among all the other groups combined. To belong rightly under this classification, however, a case should arrive there by exclusion. Rheumatism of the shoulder is altogether too easy a diagnosis. Various forms of arthritis are seen. Their features should be noted and clear-cut classification made. Improvement in treatment and benefit to the patient can thereby be greatly enhanced. Hypertrophic excrescences mechanically blocking motion can be removed successfully. Associated bursal and tendinous adhesions can be broken under general anesthesia by gentle manipulation or divided by sharp dissection when necessary. Great care must be taken to rule out a tuberculous etiology, and even with great care occasionally such cases will progress for a long period of time before their true nature is recognized. One should always suspect

the so-called infectious type of arthritis which persists for a long period with steady progression despite satisfactory treatment. An acid-fast basis may be present. It is not necessary herein to review the treatment of arthritis. We would urge to your attention, however, that arthrodesis of the tubercular shoulder is imperative. One might also well consider the availability of arthrodesis for the shoulder destroyed by any arthritic process. Relief to patient and restoration of at least partial function of the extremity may thereby be given.

5. *Pathology in the bony structures*

There are many lesions of the upper humerus and of the scapula which cause dysfunction of the shoulder and pain in this locality. The most frequent are tumors, infections, fractures, dislocations, and subluxations.

The same tumors both benign and malignant may be present in this locality as are found anywhere in the osseous system. Osteochondromas of the scapula may cause disability by their size or their impingement in motion on other structures. They may similarly be located on the upper humerus and may be called to the patient's attention either by the presence of their mass or by pain. Fibrocystic lesions in the upper humerus are very frequent and though they may cause a low grade pain, they are frequently first brought to the patient's attention by pathological fracture. Giant cell tumors are likewise frequent in this same location, are closely allied with fibrocystic bone lesions and their symptomatology and treatment are similar. Central chondromata produce low grade expansible-type pain whose cause can be determined only by x-ray.

The malignancies are represented in children by sarcoma and in the adult by metastatic carcinoma. Though sarcoma is generally accompanied by low grade pain, carcinoma may manifest itself only by pathological fracture.

Of the infectious lesions at the shoulder, osteomyelitis of the upper humeral metaphysis is much more common than suppurative arthritis of the joint.

Fractures of the surgical neck of the humerus frequently occur especially in older people without displacement and unaccompanied by the severity of symptomatology frequently seen in fractures elsewhere. They are often treated as sprains. X-ray where shoulder symptomatology exists will establish the condition present. Frequently, where such

fractures have been more severe, anatomical reconstruction is impossible and a certain loss of function of the joint is inevitable and must be accepted. Fractures of the scapular glenoid are often present and unrecognized and account for chronic shoulder subluxation and disability. Fractures of the base of the coracoid process are occasionally associated with acromio-clavicular dislocation making difficult its replacement and retention. This may promote shoulder dysfunction through lack of support.

Recurrent dislocation of the humeral head from the glenoid is often accepted by the patient and his physician as a minor disability. We feel strongly that all such recurring dislocating shoulders should be surgically repaired.

6. *Lesions due to or simulating that of tendon or bursal pathology*

All such lesions can be conveniently grouped among those due to:

A. Pathology in the tendons

B. Pathology in the bursae

C. Pathology in the adjacent bony structures

There are four flat tendons above the head of the humerus reinforcing and becoming part of the capsule. From the front to the back these are the subscapularis, the supraspinatus, the infraspinatus and the teres minor. Partial or complete laceration of one or all of these tendons may occur giving great disability. Such laceration practically always occurs when the arm is forcibly abducted as in falling on the outstretched hand. We have never seen laceration present when the patient gave a history of falling and striking on the point of the shoulder. Disability ensuing depends upon the amount of laceration present. Such laceration, except in the case of the subscapularis and possibly the teres minor tendon, do not tend to heal. This is undoubtedly because of the inability to secure good blood supply in tendons jammed between the head of the humerus and the under surface of the acromium process as well as because of the distance of separation at point of laceration.

Symptomatology will depend upon the amount of laceration present. With slight laceration of one tendon, motion of the shoulder may be complete but there will be localized tenderness at point of laceration, loss of strength in abduction and pain in lowering the arm as the greater tuberosity of the humerus passes out from under the border of the acromium process. As the amount of laceration increases, symptomatology and loss of strength will increase until finally, if two or

more tendons are lacerated, active abduction cannot be carried out beyond the point at which the greater tuberosity of the humerus impinges against the outer border of the acromium process. Passively the arm may be carried through full range of motion. If of long standing, adhesions may have developed limiting passive range of motion. Diagnosis in great part may have to rest upon history of mode of injury and suspicion on the part of the physician that such basic pathology may exist. Since laceration may be minor, or indeed, traumatism may have occurred without actual laceration, a short period of observation and physiotherapy is permissible. If symptomatology should persist beyond one or two weeks, exploration and repair are indicated. Otherwise great and permanent disability to the shoulder may result. Late repairs are extremely difficult and are unsatisfactory. Where complete laceration of the whole cuff of four tendons has taken place, or where the laceration has existed many months and a badly disabled upper extremity has resulted due to shoulder instability, shoulder fusion may restore a good degree of usefulness to the extremity. Resulting motion will be carried out by rotation of the scapula upon the thorax.

Early repair of incomplete lacerations of this muscular cuff are uniformly very satisfactory. Postoperative treatment, however, is long and a great amount of physiotherapy is necessary before satisfactory result is secured. Instances of laceration of these tendons by rough manipulation in reduction of dislocation of the shoulder or by attempts at reduction without the relaxation of good anesthesia have been encountered. May we therefore warn you in reducing dislocations of the shoulder always to use gentleness, steady traction instead of manipulation, and satisfactory anesthesia to secure perfect relaxation wherever necessary. When complete avulsion of all four tendons has occurred the cuff torn off will frequently turn downwards over the glenoid and the humeral head will lie on top of it. Hence, the shoulder will give an appearance of unusual instability with recurrent dislocation. Therefore, in cases where dislocations recur almost as rapidly as reduction is secured, this lesion should be suspected and surgical inspection and repair made. Laceration of the long head of the biceps frequently accompanies major cuff tears. It should be looked for and repaired or transplanted. Where such a condition exists, or indeed where any major laceration of these tendinous structures is present, conservative treatment can be of no advantage.

The greater the amount of laceration, the less will be the spasm, protection and pain associated with the lesion. This is so since the tendon edges lacerated will have withdrawn from any point of pressure between the humeral head and the under surface of the acromium process in complete laceration. In partial laceration, the defect in the tendinous surface will be impinged upon by the humeral head. This will cause pain, protection and loss of motion. Not only is surgical arthrodesis indicated in cases where complete avulsion of the whole cuff has occurred but also in cases where laceration of the axillary nerve is associated with damage to the tendinous cuff.

Bursal adhesions are frequently associated with small laceration but never with complete laceration of these tendons. When present they complicate the symptomatology and diagnosis.

Definite preoperative diagnosis cannot be made, a diagnosis of internal derangement being preferable. One can be reasonably certain that pathology of the tendinous structures is present but its definite extent and nature cannot be foretold. As most lacerations begin on the deep surface of the tendon and frequently do not extend completely through its thickness, a normal tendon surface may be seen on exploration when first entering the bursa. Through a small incision anterior to the supraspinatus the under surface of the tendons can be explored with a small curved clamp for laceration and repair carried out if found. In this same manner horizontal splits within the tendon substance will likewise not be overlooked.

Calcification of the subdeltoid bursa is probably always an end result of calcification of the supraspinatus or other tendons of the rotator cuff. Such calcification probably occurs in an area of degeneration resulting from previous horizontal split or localized trauma to the tendon. Needling these lesions or washing out the calcium with through and through irrigation generally gives relief. Certain of these lesions cannot be successfully washed out. On exploration such lesions have been found to be not only calcified but actually ossified. Their surgical removal has given satisfactory relief. It is likewise to be noted that recurrence of calcified lesions frequently takes place. In such instances surgical exploration, with curettage of the degenerated area has given lasting relief. If calcification of the tendinous area does not cause symptomatology enough to demand attention it frequently finally ruptures into the subdeltoid bursa. Thereupon immediate intense pain, loss of

motion and disability ensue. Washing the calcium out of such bursal lesions is very much easier than it is from the small tendinous foci, but of course, leaves the original focus behind.

Other lesions of the subacromial bursa beside calcification occur. Laceration of the floor without laceration of the underlying tendon has been seen. Symptomatology here resembles that of mild laceration of the tendinous cuff but recovery is much more rapid. Frozen shoulder or adhesive obliterative bursitis may vary in the extent of the process and the amount of symptomatology. Partial limitation of motion of the shoulder joint with extreme pain at limits of motion represents incomplete obliterative adhesions. Most of these adhesions are located about the subscapularis tendon as noted on surgical inspection. The typical cases in which the patient holds the arm rigidly to the side and refuses any attempt at motion of the shoulder joint (with extreme pain causing loss of sleep and unrelieved by physiotherapy over considerable periods of time) represent almost complete involvement of the bursa. Frequently, adhesions in both these types of cases can be broken under general anesthesia by manipulation with moderate force. Occasionally, even with extreme force, adhesions in this latter type of case cannot be so broken. In such instances we have surgically divided through the plane of the original bursa, having found it to be represented by a fibrous raphe. In these instances prolonged support of the extremity in abduction and subsequent physiotherapy for many months have given excellent results. Occasionally, continuous physiotherapy of heat, massage and gentle stretching without forcible manipulation under anesthesia will secure recovery.

Certain lesions of the bony structures have been found to give symptomatology similar to that of laceration of the tendinous structures or bursal lesions. Avulsion of the greater tuberosity of the humerus in fracture dislocation of the shoulder represents laceration of the prolongation of the tendinous cuff through the bony substance. Such fragments avulsed should be accurately replaced surgically to secure the best eventual function. More complete and more rapid recovery will follow such procedure. Occasionally, avulsion of the superior border of the greater tuberosity may be present. Similar surgical replacement is indicated. Sharp exostoses beneath the bursal floor and arising from the upper border of the greater tuberosity of the humerus may give symptomatology similar to laceration of the supraspinatus tendon. Sur-

gical removal of such exostoses by turning back the bursal floor and later replacing it has been found to give satisfactory results.

A type of case has been encountered giving the exact symptomatology of supraspinatus or cuff damage. It is due to early osteochondritic change within the humeral head. We have found this impossible to diagnose accurately preoperatively. Therefore, if at time of operation no pathology is found in the tendinous cuff, the underlying cartilage over the humeral head should be well explored for cartilaginous changes, especially near the attachment of the rotator cuff. If such osteochondritic change is massive, no surgical relief is possible aside from shoulder arthrodesis. Where small areas are involved the cartilaginous defect can be removed and satisfactory shoulder function preserved.

The diagnosis of lesions causing disability and pain about the shoulder joint depend on careful attention to history of onset or causal factor, precise localization and estimation of objective and subjective symptomatology, combined with accurate mental visualization of the anatomy of the region. Comparatively recent developments in treatment provide a much better outlook for relief of pain and reëstablishment of function in this joint.

MORELAND COMMISSION INVESTIGATION

The New York Academy of Medicine wishes to record its position at this time with specific reference to the findings of the State inquiry held by the Moreland Act Commissioner in 1943 on the administration of the Workmen's Compensation Law.

Evidence has been presented indicating that rebates from a commercial laboratory, averaging about 40 per cent of the sums paid by carrier companies have been given to some physicians referring diagnostic work to the laboratory.

It has been further indicated that physicians have loaned money to an auditor of the State Insurance Fund, possibly to ingratiate themselves and to insure approval of their charges made for professional services to claimants in compensation cases.

The intent and effect of both actions have been detrimental to professional ethics and costly to the carriers paying for such medical services. Both are in complete disaccord with the physician's admitted obligation to protect his patients personally and economically. Both involve the division of fees—condemned under the Academy's Constitution and in direct violation of a member's pledge.

The medical profession has been criticized for lack of initiative in taking preventive or punitive action in this matter. Preventive measures can depend for effectiveness only upon the creation of public sentiment for the observance of ethical regulations. This, the Academy, acting with the profession in general, has always sought to do. Punitive action can be undertaken only when valid proof of guilt can be obtained.

In order that the exact facts may be known to the Academy, its Committee on Professional Standards has held a series of hearings with those Fellows whose names have appeared in the press in connection with the matter. In the Committee's report to the Council of the Academy it was stated that while there was a basis for suspicion of guilt in some, though by no means all, of the cases examined, legally competent proof of guilt was not available to the Committee. Nevertheless, the inclusion of these names in the list of those under suspicion obligates the Academy again to emphasize the responsibility which rests upon every one of its Fellows to avoid any appearance of participation in practices which may reflect unfavorably upon the standing of themselves, the Academy, or the profession at large.

The practices referred to are in the opinion of the Academy entirely contrary to the Hippocratic Oath taken by every physician on the receipt of his medical degree; without his signed pledge to avoid such practices no candidate for Fellowship can be considered by the Academy; and on proof of guilt in such matters, a member is subject to suspension or expulsion from Fellowship.

To those Academy Fellows accused but considered innocent the following letter was sent:

Dear Doctor:

The Council of the Academy has reviewed the evidence presented to it by the Committee on Professional Standards concerning the matter of rebates recently purported to have been given or received by certain Fellows of the Academy. The Council and Committee are convinced of your innocence.

Although it is apparent that responsibility for your association with this most unfortunate publicity cannot be said to rest upon any deliberately unethical action on your part, the Council must again emphasize that Fellows of the Academy must avoid any appearance of participation in practices which may reflect unfavorably upon the standing of the Academy and the profession. It is convinced that the avoidance of suspicion of unethical practice will always necessitate the refusal to accept anything which might be construed as representing a gratuity or a commission.

Very truly yours,

To those accused and failing to establish their innocence and to those considered guilty, the following letter was sent:

Dear Doctor:

The Committee on Professional Standards has reported to the Council of the Academy in regard to its hearing on your case in relation to the findings of the Moreland Act Commissioner on the administration of the Workmen's Compensation Act.

The Council has reviewed this report. It does not intend to take or recommend action for expulsion or suspension until such time as the evidence that may be available to the State has been legally presented. It has determined, however, that in the light of the report of the Committee and the findings of the Moreland Act Commissioner, it should request your immediate resignation as a Fellow of the Academy.

Very truly yours,

The following addition has been made to the pledge signed by applicants for Fellowship in the Academy:

The undersigned hereby agrees to accept the Council of the Academy as the sole and only judge of his qualifications to remain a Fellow.

Article IX, Section 1, of the Constitution of the Academy is in process of being amended to define more fully the term "fee-splitting" and "rebating."

HERBERT B. WILCOX, *Director.*